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Health Effects of Carbon Monoxide and Ozone

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HEALTH EFFECTS OF CARBON MONOXIDE AND OZONE

July 1981

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Office of Air, Noise, and Radiation
United States Environmental Protection Agency
Ann Arbor, Michigan

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16. ABSTRACT

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INTRODUCTION

The purpose of this report is to summarize the information known about the health effects of carbon monoxide (CO) and ozone (O_3), and how that information is used by EPA to set National Ambient Air Quality Standards (NAAQS's). An additional purpose of this report is to discuss the sources most likely to contribute to high levels of CO and O_3 and how EPA control programs will reduce emissions from these sources in the future. For ease in reading, all of the information in this report is presented in question and answer format.

1. What are carbon monoxide and ozone?

Carbon monoxide (CO) and ozone (O_3) are two pollutants found in the air which can have harmful impact on public health. Carbon monoxide is a colorless, odorless, poisonous gas. Ozone is a pungent-smelling, faintly bluish, toxic form of pure oxygen. Ozone differs from molecular oxygen (O_2) in that each molecule has an additional oxygen atom which makes it more chemically and biologically reactive.

2. What are the man-made and natural sources of CO and O_3?

High concentrations of CO are largely the result of incomplete combustion of fossil fuels, especially from highway vehicles. Processes such as forest fires and the oxidation of atmospheric methane are natural sources of CO, but together they make only a small contribution to atmospheric CO levels.

Ozone is formed by chemical reactions of volatile organic compounds (VOC) -- primarily hydrocarbons -- and oxides of nitrogen (NOx) in the presence of sunlight. Ozone originates mainly from VOC and NOx emissions produced by motor vehicles, combustion of fossil fuels and industrial processes. However, a small amount of O_3 occurs naturally in the lower atmosphere from lightning discharges and as the result of downward migration from the upper atmosphere. In addition, there is evidence that vegetation emits organic vapors that may react to form O_3.

3. Why are these pollutants regulated by EPA?

Ambient levels (the concentration in the surrounding air) of CO and O_3 can be harmful to human health and the environment, especially in urban areas where industry and/or automobiles are concentrated. Congress passed the Clean Air Act to abate the growing air pollution problem. To protect the public's health and welfare, the Clean Air Act and its 1970 and 1977 Amendments charge the U.S. Environmental Protection Agency (EPA) with identifying harmful air pollutants and setting ambient standards for each air pollutant that has observed health effects. In addition, EPA is charged with the job of enforcing these standards. Research has identified CO and O_3 as air pollutants which endanger public health at high concentrations; therefore, manmade CO and O_3 are regulated by EPA.

4. How does CO affect human health?

Carbon monoxide exposure can have harmful side effects, such as those described below, depending on the concentration of ambient CO and the duration of exposure. Carbon monoxide exposure affects human health by reducing the oxygen level of the blood. Different parts of the body then suffer because the blood cannot supply them with their normal amount of oxygen. Carbon monoxide, like oxygen, can be transported by the blood from the lungs to body tissues. Hemoglobin is the substance in the blood which normally carries oxygen. If a person is exposed to CO, the hemoglobin will transport more CO and less oxygen to body tissues than normal, since hemoglobin has a higher affinity for CO than for oxygen. Hemoglobin carrying CO instead of oxygen is known as carboxyhemoglobin (COHb). A deficiency of oxygen reaching the tissues of the body, known as hypoxia, can result from elevated levels of COHb in the blood. Hypoxia is considered to be one of the possible mechanisms of CO toxicity. Another possible mechanism involves poisoning of the body's cytochrome system by CO (the cytochrome system is the cellular pathway for utilization of energy from food).

Table 1 presents the human health effects resulting from different exposures to CO, and the corresponding COHb levels. For perspective, CO levels of 15 ppm to 30 ppm (eight-hour average) are typical on bad days in major U.S. cities. Inhalation of sufficient amounts of CO can result in aggravation of symptoms in individuals with cardiovascular diseases, central nervous system/ behavioral impairment, and possibly effects on fetal growth and development. In addition, CO exposure may pose a greater health risk to persons at high altitudes, particularly visitors who are accustomed to lower altitudes (EPA, 1979a).

Healthy, non-smoking individuals commonly maintain a blood COHb level of approximately 0.4 percent from natural body sources (EPA, 1979a). Research has shown that COHb from the inhalation of CO increases the COHb level additively (NAS, 1977). However, the COHb level of an individual which results from inhalation of a given amount of CO varies somewhat according to lung capacity and other factors that depend on the individual's age, amount of exercise, and state of health. People with established cardiovascular disease (e.g., angina pectoris) and/or pulmonary disorders are most likely to be affected by CO because of reduced resistance to hypoxia. Anemic individuals and fetuses may also be particularly sensitive to CO exposure.

Cardiovascular Systems

Experimental evidence indicates that low-level acute CO exposure (15-18 ppm for 8 hours) may aggravate angina pectoris, a cardiovascular disease in which mild exercise or excitement produces symptoms of pressure and pain in the chest, by depriving cardiovascular tissue of oxygen. Angina may result in cardiovascular damage, the degree of which is unknown, and it appears to be the initial step in a series of progressively more serious symptoms that accompany cardiovascular disease and irreversible heart damage.

Increased COHb is known to dilate blood vessels of the coronary system, permitting increased blood flow (EPA, 1979a). The additional blood flow that occurs in response to the presence of COHb is apparently not sufficient to provide normal tissue oxygenation even at COHb levels as low as four percent (EPA, 1979a). Blood vessel dilation, though having some compensatory effects, could result in coronary damage or other vascular effects as the cardiovascular system is forced to function beyond its capabilities.

Research has demonstrated that chronic exposure to CO levels that are typical in many U.S. cities (15 to 30 ppm for 8 hours) aggravates angina pectoris and dilates blood vessels, both of which may lead to more serious effects. However, there are some conflicting data and more research is needed to determine whether CO exposure from polluted air contributes to incidences of heart attack or sudden deaths from coronary disease.

Fetal Abnormalities

Experimental studies of the effects of CO exposure during fetal development of animals have shown harmful health effects such as reduced birth weight, failure to gain weight at a normal rate after birth, reduced brain protein levels at birth, and reduced levels of activity during the first year after birth (EPA, 1979a). In many cases, the effects disappeared when the animals reached adulthood. However, the implications of these findings in relation to learning and social behavior of human children are clearly of concern.

Long-term exposure of pregnant animals to CO levels of 50 ppm has been shown to result in COHb levels in the fetus that often exceed the mother's levels (EPA, 1979a) Fetal hypoxia may interfere with important developmental stages. Under normal conditions a fetus may be functioning at nearly critical tissue oxygen levels, so that even moderate CO exposure could affect development. The effect of CO on human fetuses is commonly measured by studying babies born to smoking mothers, but it should be noted that cigarette smoke contains substances other than CO that may affect fetal development.

Central Nervous System/Behavioral Effects

Experimentation on low-to-medium level CO exposures and their effects on the human central nervous system show behavioral changes in sleep, alertness, and muscular coordination (EPA, 1979a). Changes in sleep patterns may be caused by reduced central nervous system activity. Alertness is an individual's ability to detect small, unpredictable changes in his environment. Experimental results have shown that COHb levels of 1.8 and 2.3 percent are sufficient to reduce human perception of small changes in light and sound, respectively. Such blood level changes may occur in persons exposed to CO levels typical of bad days in major U.S. cities. These same experiments indicated that, after a time, ability to detect small light and sound changes is partially recovered even though COHb levels remain constant or increase.

In summary, exposure to carbon monoxide causes a reduction in the oxygen level of the blood, and may hinder the cellular mechanism for conversion of food energy into forms the body can use. Harmful side effects which can result depend on the health of the person exposed and the level and duration of exposure. CO exposure can be especially harmful for persons with cardiovascular disease or anemia, and for fetuses.

TABLE 1. HEALTH EFFECTS OF VARIOUS LEVELS OF CARBOXYHEMOGLOBIN (COHb) RESULTING FROM CARBON MONOXIDE EXPOSURE

% COHb in the Blood	Approximate Ambient CO Level Required to Produce Given COHb *		Threshold Effects
	1 hr (ppm)	8 hr (ppm)	
0.3-0.7	---	---	Physiologic norm for non-smokers.
---	35	9	EPA National Primary Ambient Air Quality Standard.
2.5-3.0	55-80	15-18	Cardiac function decrements in impaired individuals; increased blood flow.
4.0-6.0	110-170	30-45	Visual impairments, decreased alertness, reduced maximal work capacity.
10.0-20.0	280-575	75-155	Slight headache, weakness, dilation of veins, central nervous system changes, general coordination problems.
20.0-30.0	575-860	155-235	Severe headaches, nausea.

* For perspective, CO levels of 15 ppm to 30 ppm (8-hour average) are typical on bad days in major U.S. cities.

SOURCE: Air Quality Criteria for Carbon Monoxide (Preprint). 1979. Environmental Criteria and Assessment Office, Office of Research and Development, U.S. EPA, Research Triangle Park, NC. EPA-600/8-79-002.

5. How does O_3 affect human health?

Ozone can impair lung function, change the cellular composition of the lungs, increase susceptibility to infectious disease, and disturb the biochemical balance of the lungs and other organs. Ozone is an irritant which causes cough, chest discomfort, and irritation of mucous membranes of the nose, throat and trachea (windpipe) at O_3 levels that are often observed in urban environments. Exercise requiring greater air volume intake also increases irritation. Table 2 lists the health effects that may occur at various levels of ozone concentration and exposure time.

Short-term exposure (one hour) to approximately 0.30 ppm of O_3 has been shown to impair lung function in healthy persons. In the majority of experimental studies in which O_3 exposure has caused changes in lung function in healthy individuals, function has returned to normal within a few hours after exposure ended. Similar O_3 exposure for individuals with respiratory illnesses such as asthma, chronic bronchitis, or emphysema may result in moderate to severe interference with normal activities. Approximately 4.0 to 5.5 million asthmatics live in urban areas within the United States (EPA, 1978a). More than 80 percent of the 500 O_3 monitoring sites measured values greater than the 0.12 ppm EPA standard during 1977 (U.S. EPA, 1978). Therefore, it is probably safe to say that 3 to 4 million people who are particularly sensitive to O_3 pollution live in areas with high O_3 levels.

Ozone inhalation can increase the probability of infectious lung disease. The exact mechanism of this effect is not well understood. Research on rabbits has shown that O_3 damages the cells responsible for destroying infectious bacteria in the lungs. Such a finding strongly suggests the extent to which O_3 can impair the body's defenses and, thus, increase the incidence of acute and chronic respiratory disease. However, similar human responses may not occur at exactly the same doses at which effects have been demonstrated in experimental animals.

TABLE 2. SUMMARY OF HUMAN HEALTH EFFECTS OF OZONE

Ozone Concentration* (ppm)	Exposure	Effect	Reference
0.02-0.05	--	Detectable odor	Stupfel, 1976
0.10	1 hour	Minor breathing difficulties	U.S. Bureau of National Affairs, 1978
0.12	1 hour		EPA National Primary Ambient Air Quality Standard
0.15-0.25	1 hour	Decreased lung function in school children, increased asthmatic attacks, irritation of respiratory tract for persons with respiratory illnesses	FR, 44:8202, 1979
0.30	1-2 hours	Impaired lung function in those exercising rigorously, cough and chest discomfort	Ferris, 1978; U.S. Bureau of National Affairs, 1978; Casarett and Doull, 1975
0.37	1-2 hours	Reduction in lung function, impaired lung function in lightly-exercising individuals	Ferris, 1978; U.S. Bureau of National Affairs, 1978
0.50	2.75 hours	Increased fragility of red blood cells, deleterious effect on cellular enzymes	Ferris, 1978
0.50	6-10 hours	Increased frequency of chromosomal aberrations	Ferris, 1978
0.75	1-2 hours	Decreased lung function (effect enhanced by exercise), dryness of nose and throat, chest discomfort, nausea, decreased work performance.	Ferris, 1978

* For perspective, ozone levels of 0.1 ppm to 0.3 ppm (1-hour average) are typical on bad summer days in many major U.S. cities.

SOURCE: Compiled by Energy and Environmental Analysis, Inc.; Arlington, VA, 1979.

6. What are EPA's air quality standards for CO and O_3 and how do these standards protect public health?

EPA National Ambient Air Quality Standards (NAAQS) for CO and O_3 are as follows:

Averaging Time	CO Primary Standards (second highest value in the year)	O_3 Primary Standards (not be exceeded, on average, more than once per year)
8 hour	9 ppm	No Standard
1 hour	35 ppm	0.12 ppm (on a daily maximum basis)

For CO, compliance with the NAAQS means that the standard is not exceeded more than one day per year in an air quality region. For ozone, compliance means that the average number of days per year above the standard, computed over a three year period, is less than or equal to one. These standards were set and are periodically reexamined according to the results of scientific and medical studies that reflect the most recent understanding of the toxicological effects of each pollutant and the potential of each pollutant to effect the health of the general public. An independent Science Advisory Board advises EPA on the standards. Each primary standard provides an adequate margin of safety because of the uncertainty in evaluating human health problems associated with low concentrations of these pollutants, and to protect especially sensitive individuals from harmful health effects.

EPA established the NAAQS for CO in 1971 and, after recently reviewing the scientific basis for the standard, proposed revisions to the primary one-hour CO standard in August 1980. EPA established the NAAQS for photochemical oxidants in 1971 and replaced it with a revised ozone standard in 1979.

The standards shown above are known as "primary" air quality standards. For ozone, there is also a "secondary" standard. The difference between the two is that the primary standard is intended to protect human health, while the secondary standard protects trees, crops and inanimate materials, which are important contributors to human welfare.

The primary and secondary standards for ozone happen to be the same. No secondary standard is required for CO because effects on trees, crops, and inanimate materials from CO have been observed only at relatively high ambient concentrations beyond those likely to occur in urban areas.

7. What are the major sources of high concentrations of CO and O_3?

Carbon monoxide from manmade sources is produced almost totally by the incomplete combustion of fossil fuels. In 1977, an estimated 102.7 million metric tons (113.2 million U.S. tons) of CO were emitted to the atmosphere from manmade sources in the United States (EPA, 1979a). Of this total, 75.2 percent can be attributed to highway transportation sources, with approximately 8 percent, or 8.3 million metric tons of CO, emitted from industrial processes. Three industries -- chemicals manufacturing, petroleum refining, and metal refining -- are the most significant industrial sources of CO emissions, a combined 7.3 million metric tons. The remaining 17 percent of nationwide CO emissions is attributable to non-highway vehicles (construction equipment, vessels, etc.), stationary source fuel combustion, solid waste, and open burning.

Ozone is not emitted directly into the air. It is a secondary pollutant, that is, a product of atmospheric chemical reactions between two precursors -- volatile organic compounds (VOC) and oxides of nitrogen (NOx) -- which, in the presence of sunlight, react to form O_3. Volatile organic compounds and NOx are emitted from both natural and manmade sources. Over the entire U.S., VOC emissions from natural sources exceed those from manmade sources. However, manmade sources tend to be geographically concentrated in urban centers so they are by far the primary cause of high concentrations of VOC and NOx. The natural sources of VOC are spread out over a much larger area and their VOC emissions are very diluted with fresh air. In addition, urban areas with ozone problems usually have ambient VOC to NOx ratios of about 10:1, which is close to the optimum ratio for ozone formation. Rural areas typically have much higher VOC to NOx ratios, due to the fact that NO_x emissions are low in these areas. Therefore, there is insufficient NO_x in rural areas to drive the photochemical reactions necessary for substantial ozone production (EPA, 1980a).

Approximately 28 million metric tons (30.8 million U.S. tons) of VOC are emitted from man-made sources in the United States each year. Most of the hydrocarbon load, estimated to make up at least two-thirds of the VOC emissions (EPA, 1980b), occurs from gasoline evaporation and motor vehicle tailpipe exhaust emissions. Other major sources of VOC, including HC, are gasoline distribution systems, chemical processing industries, and industries that use organic solvents, such as paint.

Nationwide estimates of NOx emissions attribute 21.7 million metric tons (23.9 million U.S. tons) per year (or 95 percent) to fuel combustion (EPA, 1978a). The major sources of fuel combustion and the respective NOx emissions are: highway fuels, 7.4 million metric tons per year; non-highway fuels, 2.2 million metric tons per year; non-industrial sources, 5.8 million metric tons per year.

In summary, then, while both CO and the precursors of O_3 are emitted from a variety of source types, it should be noted that the major source of both O_3 precursors and CO is motor vehicle tailpipe emissions.

8. In what areas are concentrations of CO and O_3 greatest?

Because fuel combustion in highway vehicles is the single largest source of O precursors and of CO, pollution levels tend to be greatest in urban areas with high traffic volumes. Also, basin-like geography and meteorological conditions that reduce ventilation decrease the rate at which air mixes and disperses pollutants. Table 3 presents major U.S. cities (population greater than 200,000) with the ten worst CO and O_3 levels based on the design values used in 1979 State Implementation Plans. (Design values are the pollutant measurements that are compared to the air quality standards to determine whether the standards are being met or not. Thus, they are an indicator of a city's level of a pollutant over a recent period of time.) In 1982, 39 major cities are projected not to attain the CO standard and 36 major cities are projected not to attain the O_3 standard.

TABLE 3. MAJOR U.S. CITIES (POPULATION GREATER THAN 200,000) WITH THE TEN HIGHEST CO AND O_3 DESIGN VALUES*

Cities with Highest CO Values	Cities with Highest O_3 Values
1. Bridgeport (Fairfield), CT	1. Los Angeles (South Coast Air Basin), CA
2. Los Angeles (South Coast Air Basin), CA	2. Houston, TX
3. Phoenix, AZ	3. Milwaukee, WI
4. Denver, CO	4. New York, metropolitan
5. Cleveland, OH	5. Chicago, IL
6. Albuquerque, NM	6. Detroit, MI tied
7. New York, metropolitan	6. Cleveland, OH
8. Pittsburgh, PA	7. Philadelphia, PA
9. Chicago, IL	7. Wilmington, DE
10. Fresno, CA	7. Pittsburgh, PA tied
	7. Cincinnati, OH
	7. San Diego, CA
	8. Ventura-Oxnard-Thousand Oaks, CA
	9. St. Louis, MO
	10. Allentown-Bethlehem-Easton, PA

* In 1982, 39 major cities (population greater than 200,000) are projected to exceed the national ambient air quality standard for CO and 36 are projected to exceed the O_3 standard.

SOURCE: EPA Internal Memorandum; March 26, 1980; from G.T. Helms, Chief, Control Programs Operations Branch, CPDD, to all Air Branch Chiefs, Regions I-X.

9. Under what weather conditions and at what times of day are concentrations highest?

In general, concentrations reach the highest levels when temperature inversions occur or when stagnant air masses lie over urban areas for extended periods. Inversions (which limit vertical mixing) can occur when a layer of cool, heavy air falls and is warmed through compression. When this air layer comes to rest, it is frequently of a higher temperature than the surface air layer, so that the surface air layer is trapped. This phenomenon is known as a subsidence inversion and occurs most frequently during summer months. Ground-based inversions also may trap surface air and inhibit pollutant dispersion. They occur during cold fall and winter months when the ground cools faster than the air column, creating a cool surface air layer that will not rise, mix with upper air layers, and disperse pollutants. Finally, large-scale air masses associated with high pressure weather systems may occasionally stall for several days, leading to the buildup of high pollutant concentrations.

Ozone formation requires sunlight and high temperatures (greater than 70°F). Therefore, concentrations of O_3 are higher on sunny days, especially in the summer in the early afternoon when sunlight is more direct than it is in the winter. Subsidence inversions, which are common during summer months, further aggravate the problem by preventing the normal dispersion of O_3. In the absence of an inversion, O_3 generated during daylight hours is lost by atmospheric mixing, the natural movement of air masses, and by chemical reaction, especially on ground surfaces. Nighttime levels are consequently greatly reduced. Urban O_3 levels rise again the next day as automobiles and stationary sources emit ozone precursors and sunlight promotes the O_3 producing reaction.

Carbon monoxide concentrations often follow predictable trends. In most cities, CO levels peak at seven to nine a.m., four to seven p.m., and ten p.m. to midnight. The first two peaks are due to emissions from rush-hour traffic. The midnight peak can be attributed primarily to undispersed emissions, as a result of calm wind conditions or the reversal of wind flow patterns from the daytime norm. The highest CO levels tend to occur in the fall and winter when colder ambient temperatures result in increased production of CO emissions from cars, in addition to CO emitted from other fuel-burning sources. Also, low wind speeds and ground-based inversions which occur during winter and fall result in decreased dispersion of CO emissions and contribute substantially to the occurrence of high ground-level CO concentrations.

10. What is the air quality index given on radio and TV?

The air quality index, also known as the Pollutant Standards Index (PSI), is a health-related index designed to inform the public about harmful air pollution levels (see Table 4). The PSI is based generally on the primary short-term National Ambient Air Quality Standards (NAAQS) and Federally established levels for categorizing the severity of pollution episodes. Each day, the highest pollutant level in relation to the ambient standard for that pollutant measured at any one monitoring area is reported as the PSI index. As described in Table 4, a PSI value of 100 or above signifies that health risks are present for certain segments of the population. The proportion of the population for which health risks are present increases as the PSI value rises.

11. What effects do CO and O_3 pollution have on plant life?

Carbon monoxide and O_3 both are capable of affecting plant growth. In general, O_3 is much more harmful to plant life than CO because plants are capable of absorbing and metabolizing small amounts of CO. Effects of O_3 usually are measured as changes in plant growth (stem and root), coloration, vigor, photosynthesis, respiration, or leaf injury.

Ozone is highly toxic to plant life. It has been shown to affect the permeability of cell membranes which, depending on the O_3 concentration and length of exposure, can result in effects to the entire plant. Research has determined that chronic exposure to O_3 in concentrations at or below those often found in urban areas (0.05 ppm to 0.20 ppm) causes leaf injury and reduces root and top weight in trees, shrubs, and agricultural crops (EPA, 1978a).

Botanical experimentation has found that chronic exposure to CO levels as low as 20 ppm and 24 ppm may suppress the nitrogen-fixing bacteria in root nodules of legumes or produce abnormal leaf formation in pea and bean seedlings. However, these concentrations should not be considered threshold levels for plants in general due to the lack of supporting research findings and because plants vary widely in their sensitivity to CO (EPA, 1979a). Also, food crops and forests are not generally located near areas of dense traffic, which is where these levels of CO can occur.

TABLE 4. DEFINITION OF POLLUTANT STANDARD INDEX (PSI) VALUES

PSI Index Value	Air Quality Level	Pollutant Level					Health Effect	General Health Effects	Cautionary Statements
		TSP (24-hour) ug/m^3	SO_2 (24-hour) ug/m^3	CO (8-hour) mg/m^3	O_3 (1-hour) ug/m^3	NO_2 (1-hour) ug/m^3			
500	Significant Harm	1000	2620	57.5	1200	3750			
400	Emergency	857	2100	46.0	1000	3000	Hazardous	Premature death of ill and elderly. Healthy people will experience adverse symptoms that affect their normal activity.	All persons should remain indoors, keeping windows and doors closed. All persons should minimize physical exertion and avoid traffic.
300	Warning	625	1600	34.0	800	2260	Hazardous	Premature onset of certain diseases in addition to significant aggravation of symptoms and decreased exercise tolerance in healthy persons.	Elderly and persons with existing diseases should stay indoors and avoid outdoor activity.
200	Alert	375	800	17.0	400[c]	1130	Very Unhealthful	Significant aggravation of symptoms and decreased exercise tolerance in persons with heart or lung disease, with widespread symptoms in the healthy population.	Elderly and persons with existing heart or lung disease should stay indoors and reduce physical activity.
100	NAAQS	260	365	10.0	240		Unhealthful[a]	Mild aggravation of symptoms in susceptible persons, with irritation symptoms in the healthy population.	Persons with existing heart or respiratory ailments should reduce physical exertion and outdoor activity.
50	50% of NAAQS	75[b]	80[b]	5.0	120		Moderate[a]		
0		0	0	0.0	0		Good[a]		

[a] No Index values reported at concentration levels below those specified by "Alert Level" criteria.
[b] Annual primary NAAQS.
[c] 400 ug/m^3 was used instead of the O_3 Alert Level of 200 ug/m^3.

SOURCE: U.S. Environmental Protection Agency, "Guidelines for Public Reporting of Daily Air Quality -- Pollutant Standard Index."

12. How prevalent are these effects on plant life?

The environmental effects on plants of O_3 pollution are more prevalent than the effects of CO pollution. Many cases of regional vegetational effects of O_3 have been documented. Similar reports of CO toxicity do not appear to be available.

Ozone pollution has been recognized as a chronic pollution problem only during the last 20 to 25 years. It was first noted in Southern California, where the impact appears to be most severe. Studies were made to determine the economic losses incurred in citrus groves and vineyards as a result of O_3 in the Southern California atmosphere. It was found that O_3 was responsible for reduced plant water use, reduced photosynthesis, increased leaf and fruit drop, and severe reduction of marketable fruit yield. Lemon trees and orange trees exposed to at least 0.10 ppm of O_3 over a growing season produced 32 percent and 54 percent less fruit, respectively, than an atmosphere with no O_3. Vineyards in areas where the ambient O_3 concentration often exceeded 0.25 ppm over the May through September growing season reported a 12 percent yield reduction the first year and a 61 percent yield reduction the second year (U.S. EPA, 1978a). And from 1972 through 1976, ozone-induced damage to Southern California vegetable and field crops resulted in an estimated $14.8 million per year in consumer losses (EPA, 1979b).

Various species of pine trees also are experiencing effects from O_3 pollution, especially in certain regions of the country. In the Appalachian Mountains, a disease known as emergence tipburn (which mainly affects the eastern white pine) has been attributed to O_3 levels as low as 0.12 ppm (U.S. EPA, 1978a). Emergence tipburn appears to affect only genetically sensitive trees within the species and gradually destroys them. The eastern white pine is an important pioneer tree (i.e., it often is one of the first to grow in a previously unforested area), which starts the natural ecological progression toward a fully established forest ecosystem. The destruction of these trees, then, may disrupt the balance of some forest ecosystems in the eastern United States.

Mixed pine forests in the San Bernardino Mountains east of Los Angeles and ponderosa pine in the Sequoia National Park also have sustained serious injury from chronic O_3 exposure. Mixed pine forests located on the western slopes of the southern Sierra Nevadas appear to be affected by O_3 transported from the San Joaquin Valley of California (U.S. EPA, 1978a). These trees are an important part of California's lumber industry.

It is evident, then, that secondary O_3 standards are needed to protect the quality of forest ecosystems and food crops in the U.S. and that primary CO and O_3 standards are needed to protect the health of the U.S. population.

13. What is EPA doing to help reduce levels of these pollutants?

EPA is working to carry out the Clean Air Act as amended in 1970 and 1977 to reduce air pollution and improve air quality. In response to these amendments, EPA promulgated National Ambient Air Quality Standards (NAAQS). These standards defined the principal types of air pollutants and the levels of each

that should not be exceeded for the protection of public health and welfare. Carbon monoxide and O_3 are two of the six regulated air pollutants (the others are sulfur dioxide, particulate matter, nitrogen dioxide, and lead).

According to the provisions of the 1977 Amendments to the Clean Air Act, each State is required to develop specific State Implementation Plans (SIP's) for improving air quality in areas not meeting the NAAQS and for maintaining the purity of the air in areas as clean as or cleaner than that specified by the NAAQS. EPA is assisting the States in the technical work of pollution measurement, planning, and control so that they may develop and carry out their implementation plans. EPA also distributes Federal funds appropriated by Congress to the States for use in achieving air quality goals.

Besides setting and enforcing air quality standards, EPA also is responsible for establishing pollution emission limits for automobiles. To enforce these emission standards, EPA tests prototype models of all new cars and trucks to ensure that they comply with the legal standards, performs assembly line tests and orders recalls when defective emission control components are identified through in-use testing. And under EPA's guidance, state are implementing inspection and maintenance (I/M) programs to reduce levels of in-use emissions from motor vehicles.

Another EPA program mandated by the Clean Air Act is the New Source Performance Standards (NSPS) for stationary sources, which are emission limits for certain source categories (e.g., power plants) determined to contribute significantly to air pollution levels.

14. How much progress has been made?

Progress has been made both in the development of programs to clean the air (planning) and in actual reductions in ambient pollutant concentrations (implementation). For example, State Implementation Plans (SIP's) have been developed by all States. These plans include a definite timetable for a step-by-step attainment of National Ambient Air Quality Standards (NAAQS). Also included in the SIP's of those States granted extensions from 1982 to 1987 for demonstrating attainment with O_3 and/or CO standards are plans for motor vehicle inspection and maintenance, and programs to improve mass transportation and reduce personal vehicle use in urban areas, in order to reduce automobile exhaust emissions.

In general, ambient levels of CO are lower now than in the recent past and are continuing to decline. Between 1972 and 1977, approximately 80 percent of the monitoring sites in the United States showed long-term improvements in CO levels (EPA, 1978b). This trend is fairly consistent throughout the United States. On the other hand, O_3 trends are not as encouraging. About one-third of all monitoring sites throughout the country have noted increasing O_3 levels from 1972 to 1977 (EPA, 1978b). And in the period from 1974 to 1978, there were no substantial changes in the yearly average of daily maximum ozone levels for a collection of 51 sites studied by the President's Council on Environmental Quality (CEQ, 1980). However, ozone monitoring in Los Angeles, where the problem is most severe, has indicated substantial long-term reductions in ambient levels.

15. What are the legal deadlines for attainment of the standards?

Provisions of the Clean Air Act Amendments of 1970 originally established July 1975 as the deadline for compliance with the National Ambient Air Quality Standards (NAAQS). This deadline was found to be unachievable and was extended to 1982 by the 1977 Amendments to the Clean Air Act. For some States, the deadline was further extended to 1987 for CO and O_3 because of the demonstrated difficulty in reducing current emission levels, especially of motor vehicles, within a relatively short time frame.

16. What is EPA doing to help ensure attainment of the standards?

EPA has a number of continuing programs to ensure that CO and O_3 NAAQS's are attained and maintained. For mobile sources, the Federal Motor Vehicle Control Program provides for an increasingly stringent set of emission standards designed to control emissions from cars, trucks, buses, and motorcycles. As new vehicles with low emission levels replace older vehicles with high emissions, the total pollutant burden from motor vehicles should be lessened. However, emissions will continue to deteriorate as individual vehicles age. In addition, total miles traveled by passenger vehicles increased during the decade from 891 billion miles per year in 1970 to 1.1 trillion miles per year in 1978 (MVMA, 1980); this meant that emission reductions in the 1970's were at least partially offset by increased vehicle usage and this could also happen in the 1980's. Population and economic growth have also led to increases in emissions from sources other than motor vehicles. Therefore, in order to further reduce motor vehicle emissions, Congress required in the Clean Air Act Amendments of 1977 that inspection and maintenance programs be implemented if State Implementation Plans for O_3 and/or CO cannot demonstrate attainment of the NAAQS by 1982. I/M programs will help maintain the efficiency of automotive emission control systems by requiring repair of vehicles which do not pass a tailpipe emission test. Transportation control measures are another option available to States and local areas for reducing mobile source pollution. Examples of transportation control measures include carpool/vanpools, express bus lanes, park-and-ride lots, and parking surcharges.

Controls on stationary sources are dependent on whether the source is in an attainment or a nonattainment area and whether the source is to be constructed or already exists. Some new sources have emission limits dictated by New Source Performance Standards (NSPS's) if they are in a category of sources determined to contribute significantly to air pollution levels. New sources in nonattainment areas are required to control to the lowest achievable emission rate, while new sources in attainment areas must use the best available control technology. Existing sources in nonattainment areas are required to reduce emissions by adopting at a minimum reasonably available control technology.

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REFERENCES

Casarett, L.J., and Doull, J. 1975. Toxicology, The Basic Science of Poisons. New York: MacMillian.

Ferris, B.J. 1978. "Health Effects of Exposure to Low Levels of Regulated Air Pollutants," Journal of Air Pollution Control Association 28(5):482-497.

National Academy of Sciences (NAS). 1977. Carbon Monoxide. Prepared by the Committee on Medical and Biologic Effects of Environmental Pollutants, National Research Council.

Stupfel, M. 1976. "Recent Advances in Investigations of Toxicity of Automotive Exhaust," Environmental Health Perspectives 17:253-285.

U.S. Bureau of National Affairs. 1978. "Criteria for Ozone and Other Photochemical Oxidants," Environmental Reporter, Federal Laws Index BNA:31:2151-31:2162.

U.S. Environmental Protection Agency (EPA). 1978a. Air Quality Criteria for Ozone and Other Photochemical Oxidants. EPA-600/8-78-004.

U.S. EPA. 1978b. National Air Quality, Monitoring and Emissions Trends Report, 1977. EPA-450/2-79-022.

U.S. EPA. 1979a. Air Quality Criteria for Carbon Monoxide (Preprint). EPA-600/8-79-022.

U.S. EPA. 1979b. Methods Development for Assessing Air Pollution Control Benefits, Vol. III. EPA-600/5-79-001c.

U.S. Government Printing Office. 1979. Federal Register 44:8202.

U.S. EPA. 1980a. Natural Sources of Ozone: Their Origin and Their Effect on Air Quality. EPA-AA-IMS/AQ-80-2.

U.S. EPA. 1980b. Review of Criteria for Vapor-Phase Hydrocarbons. EPA-600/8-80-045. RTP, N.C.

EPA Internal Memorandum; March 26, 1980, from G.T. Helms, Chief, Control Programs Operations Branch, CPDD, to all Air Branch Chiefs, Regions I-X.

Council on Environmental Quality. 1980. Environmental Quality - 1980. The Eleventh Annual Report of the Council on Environmental Quality.

Motor Vehicle Manufacturers Association of the U.S. Inc. (MVMA). MVMA Motor Vehicle Facts and Figures '80. Detroit, Michigan.